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Effects of neurofeedback training on objective and subjective measures of attention deficit hyperactivity disorder in children

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EFFECTS OF NEUROFEEDBACK TRAINING ON OBJECTIVE AND SUBJECTIVE MEASURES OF ATTENTION DEFICIT HYPERACTIVITY DISORDER IN CHILDREN

A Thesis

Presented to the

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ABSTRACT

Attention Deficit Hyperactivity Disorder (ADHD) is a pervasive disorder characterized by inattention, hyperactivity and impulsivity diagnosed primarily in children, that often continues into adulthood. Diagnosis is best accomplished using a checklist based on DSM-IV criteria and the Test of Variables of Attention (T.O.V.A.), an objective tool with 4 measures: Attention, Impulsivity, Reaction Time, and Variability. Psychostimulants are the most common treatment for ADHD but symptom reduction is only temporary, and there are undesirable side effects. Characteristic EEG patterns are associated with ADHD which can be controlled by Neurofeedback. This study examines the effects of Neurofeedback on 3 subscales (Inattention, Hyperactivity and Impulsivity) of the Oaks checklist for ADHD, and on the T.O.V.A.'s 4 subscales (see above). The study compared 2 treatment conditions, using a reverse order design. One condition was Neurofeedback reinforcement of sustained production of mid-frequency EEG (SMR/beta, 12-18 Hz) activity accompanied by inhibition of low-frequency EEG (theta, 4-7 Hz) activity. The other condition was ThinkFast a manually operated computer game designed to enhance mental processing. Participants were 3 females and 17 males, ages 5 to 15 years, divided into two groups. Group 1 (n = 10) received 20 sessions of Neurofeedback, followed by 20 sessions of ThinkFast.

Group 2 (n = 10) received 20 sessions of ThinkFast followed by 20 sessions of Neurofeedback. The T.O.V.A. and the Oaks were administered before and after each 20 sessions of treatment. Results showed that children were able to maintain criteria levels of 12-18 Hz EEG activity within training sessions and across training sessions. Within group improvement was found on T.O.V.A. subscales for Impulsivity in Group 1 after Neurofeedback. Within group improvement was found on the Oaks subscales for Hyperactivity and Impulsivity in Group 1 after Neurofeedback. Within group improvement was found on the Oaks subscales for Inattention, Hyperactivity and Impulsivity in Group 2 after Neurofeedback. Large group differences on initial T.O.V.A. and Oaks measures and within group variability contributed to conflicting results. An unexpected finding was that ThinkFast may enhance the effects of Neurofeedback when used to treat ADHD. Overall, results suggest that Neurofeedback should continue to be subject to future study as an effective treatment for ADHD.

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INTRODUCTION

Attention Deficit Hyperactivity Disorder (ADHD) is described in The Diagnostic and Statistical Manual of Mental Disorders - IV (American Psychiatric Association, 1995) as a "behavioral syndrome characterized by a persistent pattern of inattention and/or hyperactivity/impulsivity that is more frequent and severe than is typically observed in individuals at a comparable level of development." Some evidence of ADHD is typically manifest between the ages of two and seven. Recent studies have revealed that about 80% of these children continue to be affected through adolescence into adulthood with age-appropriate modification of symptoms (Wender, 1995). Impairment from these symptoms affects the ability of the individual to function optimally in a variety of settings, such as home, school, social activities, and sports. Children with this behavioral syndrome are frequently found to be functioning below appropriate developmental levels (Wender, 1995). Social and family relationships are often characterized by resentment and antagonism with associated low self-esteem in the ADHD child because their symptomatic status is so variable that troublesome behavior is often interpreted by others as being willful misconduct (Wender, 1995).

History

The impulsive, disruptive behavior pattern associated with ADHD was first documented by George Still, in the British medical journal *Lancet* in 1905, who characterized it as a disorder of "moral behavior associated with wonton destructiveness" (Lubar, 1997b). In the 1920's, following a severe influenza epidemic in 1918, similar behavior was noted in a number of children who had had influenza-related encephalitis (Strauss and Lehtinen, 1947). During the 1930's and 1940's researchers identified a series of disorders of children which all seemed to involve some type of minimal brain dysfunction syndrome (Strauss and Lehtinen, 1947). Often a history of brain injury, toxic reaction to heavy metals, perinatal complications, or genetic factors were found to be associated with varying degrees of behavioral dysfunctions, such as hyperactivity and poor ability to pay attention (Strauss and Lehtinen, 1947). Strauss and Lehtinen (1947) developed the concept of Minimal Brain Dysfunction (MBD) in an attempt to combine these symptoms into one main disorder.

By the 1970's MBD had been separated into more specific disorders: Hyperkinetic Disorder, disorders of attention, specific learning disabilities, and disorders of conduct. In 1987, the DSM III-R classified the condition as Attention Deficit Disorder (ADD) with four subcategories, which included ADD with, and without

Hyperactivity, conduct disorders (CD) and specific learning disabilities (SLD). The current DSM-IV now refers to the general syndrome as Attention Deficit - Hyperactivity Disorder (ADHD) which is further categorized into three subtypes according to criteria designed to identify specific behaviors associated with either inattention, or hyperactivity-impulsivity: ADHD, combined type; ADHD predominately inattentive type; and ADHD predominately hyperactive-impulsive type.

ADHD Characteristics

ADHD combined type, includes features of both inattention and hyperactivity-impulsivity. Inattention may be manifested by failure to attend to details, making careless mistakes, messy or careless work, difficulty staying on task or sustaining attention, frequently appearing to be daydreaming or not listening (DSM-IV, 1994). Other features include difficulty organizing activities and a tendency to be easily distracted and forgetful, often losing track of items needed for an activity. For example, forgetting to bring home books needed for homework (DSM-IV, 1994). Resnick, Hamer and Goldberg (1988) identified four other features often associated with ADHD. Difficulty chaining commands, that is, remembering more than one or two commands at a time; difficulty benefiting from one's own experience; school grades which are variable across grading periods; and

difficulty completing school assignments (Resnick, Hamer and Goldberg, 1988).

Symptoms of hyperactivity-impulsivity are primarily characterized by excessive physical activity that is inappropriate for the situation (running about or climbing), interrupting others, squirming or fidgeting, or being constantly on the go (DSM-IV, 1994). Hyperactive behaviors also include difficulty playing quietly and tending to make excessive noises during quiet activities (DSM-IV, 1994). Impulsive behaviors include impatience, speaking at inappropriate times, intruding on others, restlessness, and a low frustration tolerance level (Lee, 1991).

Impulsivity and lack of thinking through the consequences of an action, sometimes combines with gross motor difficulties such as clumsiness or stumbling and often results in accidents. Fine motor difficulties, for example, poor or slow handwriting are also frequently observed in ADHD (Silver, 1988).

Silver, (1988) also cites the co-existence, in many cases, of visual and auditory figure-ground disturbances. Visual figure-ground difficulties are those which involve problems focusing on the appropriate figure as separate from other background stimuli demonstrated by skipping words or jumping lines when reading. Auditory figure-ground problems are demonstrated by similar difficulties

separating appropriate sounds from background noise. In some cases, the ability to integrate information in the form of sequencing is affected (as in reversing numbers or letters, or mixing the sequence of thoughts or events in a story. Another area often affected is abstraction of information, the ability to infer meaning other than the literal meaning to words or phrases based on subtle clues in the context. Any or all of these difficulties may underlie problems of reading comprehension, memory and expressive language frequently associated with ADHD. These difficulties are usually associated with the poor academic performance which often accompanies ADHD (Silver, 1988).

Although the diagnostic characteristics of ADHD do not include oppositional and aggressive behaviors, a high percentage of young children with ADHD display these characteristics. Disobedience, talking back, and frequent fighting are characteristic of oppositional-defiant disorder (ODD) or conduct disorder (CD) which have a high rate of comorbidity with ADHD (DSM-IV, 1994; Greene, Biederman, Faraone, and Sienna, 1997; Wender, 1995). In adolescence these behaviors seem to lead to problems with drug or alcohol abuse and encounters with the law (Lomas, 1995; Wender, 1995), and one might expect continued and possibly more severe problems in adulthood.

Prevalence, Age and Gender Features

The DSM-IV estimates that ADHD occurs in 3% - 5% of school age children. It is also known to occur in various cultures outside the United States (DSM-IV, 1994).

Behaviors characteristic of ADHD vary somewhat according to developmental level and gender. Preschool-age children with ADHD are more difficult to diagnose because there are fewer demands on them for sustained attention. However they may be unable to sit still long enough to look at a picture book with an adult, or be constantly moving and difficult to contain (DSM-IV, 1994).

According to Silver (1988), school-age ADHD children frequently manifest disruptive classroom behaviors such as non-compliance, being out-of seat, self-talk and self-stimulation in the form of playing with objects or similar behaviors. Academic performance often ranges from poor to variable with grades ranging from A to F in a single subject (Buchoff, 1990; Carlson, Pelham, Milich, Hoza, 1993; Silver, 1988). Impulsive behaviors may lead to difficulty following rules at school and at home, which may continue and even increase during adolescence. These behaviors often carry over into adulthood (Barkley, Guevremont, Anastopoulos, DuPaul and Shelton, 1993; Carlson, et al., 1993; Silver, 1988; Wender, 1997).

ADHD symptoms in adolescents may include academic underachievement due to failure to finish work, failure to

sustain attention, disorganization, and not listening to or following directions. Symptoms also include immaturity expressed by excessive silliness or fooling around and overreaction to teasing or normal peer interactions (Barkley, et al., 1993; DSM-IV, 1994, McGee and Share, 1988).

The incidence of ADHD in males versus females has been found to range from 3:1 to 9:1 in favor of males (Brown, Madan-Swain and Baldwin, 1991; Buchoff, 1990). The ways in which ADHD is manifest differs somewhat according to gender. Brown, et al. (1991), found that girls were more likely to be held back in school and showed greater impairment on visual-spatial measures than boys. There was a trend for girls to be diagnosed at an older age than boys, with impairment increasing with age across a wider span of measures including cognitive functioning, academic achievement and peer relations. Boys tended to be more aggressive than girls, and to be rated by teachers as unpopular at an earlier age than girls (Brown, et al., 1991; Gaub, 1997). With age boys improved on neurocognitive measures but experienced greater difficulties in reading comprehension and therefore in academic achievement (Brown, et al., 1991; Silver, 1988).

According to Wender (1995), about 80% of children with ADHD do not outgrow it and it persists to some degree, into adulthood. The DSM-IV recognizes the continuation of ADHD

into adulthood although the symptoms may be attenuated. Adults with ADHD generally retain attenuated symptoms but often they are enough to cause functional impairment (Wender, 1997; Lomas & Gartside, 1997; Toone and van der Linden 1997).

Adults may experience subjective feelings of restlessness or subdued fidgeting. They may be unable to engage in a focused activity such as reading; they may behave in an absentminded manner or experience extreme irritation when having to wait in lines or traffic. They may also experience frequent mood swings, disorganization (i.e., switching form task to task haphazardly) and have difficulty solving problems or managing time (Barkley, et al., 1993; DSM-IV, 1994; Hunt, 1997; Lomas, 1995; Ratey, Hallowell, and Miller, 1997; Wender, 1997).

According to Lomas (1995), many ADHD adults have a low tolerance for stress, often displaying constant irritation or frequent explosive outbursts. They often make spur-of-the-moment decisions based on little information and with little reflection on expected consequences. Relationships are often begun and ended abruptly. Adults with ADHD frequently seem to be accident prone because of clumsiness and poor sense of direction (Barkley, et al. 1993; Hunt, 1997; Lomas, 1995; Silver, 1988).

The evidence is mounting that ADHD not only continues into adulthood, but may be a significant factor in major

psychosocial difficulties suffered by those afflicted with it. ADHD in adulthood is often associated with increased problems with job and family, and encounters with the law (Barkley, et al., 1993; Bhandary, 1997; Jerome and Segal, 1997; Milberger, Biederman, Faraone, and Chen, L., 1997; Ratey, et al., 1997). One study found that licensed drivers with ADHD, between the ages of 16 and 22 had more automobile accidents, and more bodily injuries associated with those accidents. They were also found to be at fault for more of the accidents than a control group of non-ADHD, licensed drivers in the same age range. This same group of drivers with ADHD was also more likely to be cited for speeding and other traffic violations (Barkley, et al., 1993). Milberger, et al. (1997), and Wilens, Biederman and Mick (1998) note a positive relationship between ADHD and substance abuse in adulthood.

Some authors believe there may be some danger in overdiagnosing ADHD in adults, but Lomas (1995) points out that it may be more tragic to not consider the possibility of ADHD in adults, and risk leaving it undetected or untreated. He cites an example of a colleague who spent years in personal analysis and postanalytic therapy before he was finally diagnosed with ADHD and treated with good results. Lomas and Gartside (1997) found a significant percentage of homeless veterans screened positive for ADHD.

Developmental Implications

The developmental implications of ADHD are very important. If a child is unable to perform optimally in an academic setting or a social setting there is a strong possibility that they will remain developmentally behind their peers into adulthood. Unless ADHD is recognized and treated the social and family problems are likely to become worse (Silver, 1988). The sense of inadequacy and failure that accompanies ADHD is potentially crippling from an emotional perspective as well. These children often experience a sense of helplessness and negative attributional style that is carried with them for the rest of their lives (Carlson, et al., 1993). They frequently have a poor self-image and often withdraw, or strike out impulsively from frustration with their inability to perform at the level expected of them by parents, teachers, peers and especially themselves. They may suffer from depression, and/or anxiety, and may internalize stressful feelings. This may lead to real physical symptoms such as headaches or stomachaches (Silver, 1988).

Assessment and Diagnosis

Psychological and developmental evaluation and assessment are important in providing a complete clinical profile. There is a wide range of tools to assess behaviors when evaluating a child for ADHD. One commonly used tool is the Conners Parent-Teacher questionnaire which

consists of a 28 question teacher section, and a 48 question parent section (Conners, 1973). These are most useful for younger children who are frequently in the company of either the parents or the teacher for a large part of the day. Conners (1973) also published an adolescent self-report, the ADD/H Adolescent Self-Report Scale (ADD/HSRS), which Wender (1995) suggests is more valuable for assessing adolescents than the Conners Parent-Teacher questionnaire. Visual-spatial memory tests such as the Bender Gestalt test and auditory memory measures such as the Detroit Tests of Learning Aptitude may be useful in assessing visual and auditory processing. Some measure of academic achievement to detect specific learning disabilities is also helpful (J.Horn, personal communication, December, 1995). Examples of academic achievement tests include the Wechsler Intelligence Scale for Children Revised (WISC-R), or the Wide Range Achievement Test (WRAT).

An additional objective measure of attention that is frequently used is the Test of Variables of Attention (T.O.V.A., developed by Greenberg (1987). The T.O.V.A. is a continuous performance test (CPT) which measures response time. It analyzes response times for errors of omission (as a measure of inattention); errors of commission (as a measure of impulsivity); the mean reaction time, and the variability in response times (as a measure of

consistency). The T.O.V.A is a non-language-based, 22 ½ minute, fixed-interval, computerized test designed to be effective for use over a wide range of participants. It is able to identify the degree of impairment in attention and impulsivity that tend to be associated with ADHD. It is a useful tool to help differentiate between normal functioning individuals and those with attention or impulsivity problems. By itself, the T.O.V.A. does not diagnose ADHD, but it is helpful in identifying those related characteristics, and should be considered in combination with other assessment tools (Greenberg and Dupuy, 1993).

A good medical/developmental history and physical examination in addition to basic psychological evaluation are important in order to eliminate any underlying medical problems and identify developmental risk factors that may be associated with ADHD (Greenberg, 1987). High risk factors such as family history of ADHD or substance abuse, birth trauma or minor head injuries can be identified while obtaining the history. The physician's traditional role has been to perform laboratory or other tests to rule out possible medical reasons for ADHD symptoms such as; vision or hearing problems, allergies, anemia, hyperthyroidism, hypoglycemia, substance abuse or medication reactions (Sears and Thompson, 1997; Silver, 1988).

Etiology

The cause of ADHD is still unclear, however there appears to be more support for a neurological basis than a psychosocial basis. Various theories include: genetic predisposition, prenatal and birth related difficulties resulting in subtle brain damage, mild head injuries, neurochemical imbalances, delayed neurological development, neurosensitivities to dietary and other environmental substances, and nutrient deficiencies (Dalton, 1996; DSM-IV, 1994; Feingold, 1974; Lomas, 1995; Lubar 1997b; Resnick 1988; Silver, 1988. One theory submits that ADHD is the *result* of learning disabilities, instead of vice versa (McGee and Share, 1988).

In addition to sometimes being regarded as a maturational problem of the nervous system, it is speculated that ADHD may be due to a neurotransmitter deficiency. Evidence to that theory is presented by Shekim, Sinclair, Glaser, Horwitz, Javaid and Bylund (1987), and Wender and Reimherr (1990) which suggests an association between decreased levels of dopamine and/or norepinephrine, and ADHD. Other data exists which suggest that dopaminergic activity is lower in adults with ADHD (Lubar, 1997b). According to Lubar (1997b), a dopamine deficit exists in the communication system between the brainstem and the prefrontal and central cortical areas often accompanied by excess norepinephrine.

Treatment and Management of ADHD

A number of therapies exist for ADHD including, but not limited to: cognitive-behavioral therapy (Gomez, 1991), dietary therapy (Dalton, 1996; Feingold, 1974), pharmacotherapy (Gomez, 1991, Greenhill, 1992, Nathan, 1992; Potashkin and Beckles, 1990), and biofeedback therapy (Lee, 1991; Lubar, 1991; Lubar, 1997b; Lubar and Shouse, 1976b; Lubar, Swartwood, Swartwood and O'Donnell, 1995; Tansey, 1991; Tansey, 1993). In most cases a comprehensive approach tailored to the appropriate developmental level is most effective (Lubar, 1997b; Nathan, 1992). It is also important to educate the family about ADHD. The structure and dynamics of family and classroom functioning provide a framework and background for more definitive therapies (Buchoff, 1990; Carlson et al., 1993; Most of these therapies take a period of days or weeks to demonstrate effectiveness (Gomez, 1992; Nathan, 1992). Even pharmacotherapy is accompanied by the warning that it may take four to six weeks to show an effect (PDR, 1998). During that time, a home and classroom management program may be able to reduce the effects of behavioral problems (Buchoff, 1990; Gomez, 1992; Nathan, 1992).

Family and teachers need to know what can realistically be expected from the child with ADHD. They also need to understand the nature of the disorder underlying many of the child's troublesome behaviors

(Silver, 1988). Structural and behavioral therapies can then be applied in both the family setting and the classroom setting. If the child is experiencing academic difficulties specific education programs may also be needed (Buchoff, 1990; Gomez, 1992; Nathan, 1992). In some cases, parents may need to initiate the process for obtaining educational assistance. In any case, parents need to become an active part of the team effort along with the teacher(s) and the child himself, in the process of academic remediation and behavior modification (Buchoff, 1990; Gomez, 1992; Nathan, 1992).

It is important for parents of the child with ADHD to learn about the nature of the disorder and how it affects the child's ability to sustain attention, and control impulsive and hyperactive behaviors (Barkley, 1990; Buchoff, 1990; Silver, 1988). It is also important for parents to understand related problems such as anger management, aggression and anxiety. Parents need to learn skills of child behavior management in order to help the child with ADHD to learn self-control skills (Barkley, 1987; Buchoff, 1990; Nathan, 1992; Silver, 1988).

Parental reinforcements need to be immediate and consistent, whether it is rewards or punishment. Rules and instructions should be stated as simple direct imperatives in a neutral tone of voice (Buchoff, 1990). Anticipating problem situations and teaching the child appropriate ways

to maintain self-control beforehand helps increase the ability of the child with ADHD to function effectively (Barkley, 1987; Buchoff, 1990). Finally, parental modeling of appropriate interaction patterns with other family members is a better way to teach any child with ADHD than giving complicated instructions and lectures on the why and wherefore of social convention (Barkley, 1987; Buchoff, 1990).

In the classroom, one overriding principle should be to convey to children with ADHD that the teacher is personally interested in helping, and cares about them. Children with ADHD require almost constant supervision since they cannot keep themselves organized (Buchoff, 1990). Teachers can help the child and themselves by simplifying the child's environment, helping establish routines, and providing assistance in organizing time and space (Buchoff, 1990). Directions should be given only after getting the child's attention by making eye contact, and then they should be clear and simple (Buchoff, 1990).

Within the past few decades, a variety of dietary theories and approaches have developed in an attempt to find a nutritional correlation with ADHD. Feingold (1974) was among the first to postulate a relationship between foods and ADHD. His approach focused on exclusion of artificial food additives such as colorings, flavorings and preservatives; also on restriction of dietary sugar intake

and on identification and avoidance of food allergens (Feingold, 1974). The Feingold diet became popular in the late 1970's and still has some followers today. According to Grossman (1982), reports by Dr. Keith Conners (1980), and The National Advisory Committee on Hyperkenesis and Food Additives (1980), among others, concluded that there may be a small percentage, of children with ADHD who show sensitivity to food additives (Grossman, 1982).

Behavior modification programs and cognitive therapy programs have had limited success as primary treatment modalities for ADHD. These programs have been most successful when psychopharmaceuticals are given at the same time (Gomez and Cole, 1991; Nathan, 1992; Pelham, 1993; Woltersdorf, 1992).

It is well known that the most effective and widely used treatment for ADHD up to this time has been psychopharmaceuticals, primarily stimulants. ADHD appears to be the result of a neurological abnormality in the brain (Chabot, Merkin, Wood, Davenport, and Serfontein, 1996; Lahat, Avital, Barr, Berkovitch, Arlazoroff and Aladjem, 1995; Lubar, 1997a; Suffin and Emory, 1995) which certain types of psychopharmaceuticals are capable of correcting in some cases, and to some extent (Greenberg, 1987; Nathan, 1992, Potashkin and Beckles, 1990).

Stimulants have been found relatively safe in about 70% of children with ADHD. The most common stimulants used

are methylphenidate (MPH; or Ritalin), dextroamphetamine (Dexedrine), and pemoline (Cylert), with a greater number receiving MPH than dextroamphetamine or pemoline (Wender and Reimherr, 1990; Greenhill, 1992; Steingard, Biederman, Spencer, Wilens and Gonzalez, 1992; Pelham, 1993). Anti-depressants such as the tricyclics and monoamine oxidase inhibitors (MAO-I) have also been used with limited success (Greenhill, 1992; Pelham, 1993). However, there have been serious side effects reported with these drugs (Greenhill, 1992; Pelham, 1993). Recently bupropion has been explored as a relatively safe and effective medication for treating adults with ADHD (Wender and Reimherr, 1990; Greenhill, 1992). However, bupropion has potential for causing seizures in rare cases, so individuals must be carefully monitored and alerted to this possibility (Physician's Desk Reference, 1998). The primary disadvantage of psychostimulants is short duration of effects, four to eight hours in most cases; and once medication's effects wear off the symptoms of ADHD frequently return full strength (Barkley, 1990; Lubar, 1997b; Pelham, 1993).

Treatment of ADHD with medication has other drawbacks. The child must be willing to take the medication and in some cases, especially as the child becomes older, they will not cooperate with a medication treatment program. Medication must be given or supervised by the parent which

is sometimes difficult when the medication needs to be given mid-day as well as early morning (Pelham, 1993).

There are many side effects associated with all these medications, some less tolerable than others. The most common side effects of Ritalin include nervousness and insomnia, suppression of growth (weight and/or height), and aggravation of tic disorders (Greenhill, 1992; Pelham, 1993). The Physician's Desk Reference (PDR, 1998) lists other common reactions which include hypersensitivity, loss of appetite, abdominal pain, weight loss, visual disturbances and tachycardia. Common side effects of Cylert include those given above for Ritalin plus liver dysfunction and convulsive seizures (Greenhill, 1992; Pelham, 1993; PDR, 1998). Dexedrine's common side effects are the same as those of Ritalin with the addition of other gastrointestinal disturbances plus anorexia, dizziness and euphoria (Greenhill, 1992; PDR, 1998).

Considering the side effects and possible toxicity associated with long term use of medications for ADHD treatment and their short term effects on ADHD symptoms, a non-invasive treatment which could produce positive changes in the underlying neurophysiology would be very welcome. If this same treatment offered the possibility of long-term effects even after treatment is stopped it would be an even more attractive alternative to long term use of medication.

Several methods of biofeedback have been the subject of studies involving treatment of ADHD. Among these methods are: skin temperature, galvanic skin response (GSR), electromyographic (EMG), and electroencephalographic (EEG) biofeedback, with varied levels of effectiveness (Lee, 1991; Linden, Habib and Radojevic, 1992; Lubar and Shouse, 1976a; Lubar, 1991, Lubar, et al., 1995; Lubar, 1997b; Mulholland, 1995; Othmer, 1992; Potashkin and Beckles, 1990; Rossiter and La Vaque, 1995; Shouse and Lubar, 1979; Tansey, 1991; Tansey, 1993).

Studies of EMG and EEG biofeedback have shown a high degree of effectiveness, especially in combination with other treatment modalities such as relaxation training, visual imagery, behavior modification techniques and stimulant medication (Lee, 1991). EMG biofeedback gives the participant feedback on the state of their muscle tension or relaxation. The goal is to attain awareness and control or muscle tension associated with hyperactivity-impulsivity which may enable the individual to gain control of their motor behavior (Mulholland, 1995). GSR and skin temperature biofeedback are also designed to increase one's ability to relax thereby decreasing motor activity (Mulholland, 1995).

EEG biofeedback, more recently termed Neurofeedback, gives feedback on one's mental state, training the individual to attain a relaxed but focused state of mind.

Neurofeedback procedures reward production of mid-range frequencies of brainwaves associated with relaxed alertness and at the same time inhibit excess production of low frequency brainwaves associated with a less alert mental state, often referred to as a "day-dreaming state", or feeling "spacey". Neurofeedback procedures may also be used to inhibit excess production of high frequency brainwaves associated with hypervigilance, anxiety or underlying tension (Lubar & Shouse, 1976; Othmer and Othmer, 1992). This treatment is supported by evidence of disturbances in neurotransmitter activity and neurometric studies which found abnormal EEG activity in prefrontal and central brain areas of children with ADHD (Chabot, et al., 1996; Fried, 1993; Janzen, Graap, Stephenson, Marshall and Fitzsimmons, 1995; Lahat, et al.., 1995; Lubar, 1991; Lubar, 1997a; Lubar and Shouse, 1976a; Satterfield, Lesser, Saul and Cantwell, 1973; Shekim, et al.., 1987; Suffin and Emory, 1995).

Neurofeedback enables the individual with ADHD to modify brainwave activity in the direction of normalization, or homeostasis, with corresponding ability to manage behavior in ways that may result in better academic performance and better social relationships (Lubar, 1997b).

Neurophysiological Features of ADHD

Two early papers were published by Satterfield and his colleagues (1971, 1973), proposing that hyperkinetic behavior in children was due to a disturbance in state of arousal, and because of this they were easily habituated to sensory stimulation and therefore constantly sought stimulation. They also suggested that there might be an abnormality in adrenergic neurotransmitter production and utilization resulting in a problem with reticular activation (Satterfield and Dawson, 1971; Satterfield, Lesser, Saul and Cantwell, 1973).

More recent work suggests that the primary symptoms of ADHD are really secondary manifestations of an underlying neurological disorder. Evidence for this is decreased cortical arousal associated with decreased noradrenergic activity and increased slow wave, or theta (4-8 hertz [Hz]) activity in frontal and central cortical regions, and decreased glucose metabolism in both frontal cortical and certain subcortical regions; Janzen, et al., 1995; Lubar, 1991; Lubar, et al., 1995; Zametkin, Nordahl, Gross, King, Semple, Rumsey, Hamburger and Cohen, 1990). Lubar (1991) suggests that an even better indicator of ADHD is the ratio of theta (4-8 Hz) activity to beta (16-20 Hz) activity in the frontal cortex. Janzen, et al. (1995) found consistently larger ratios of theta activity to beta activity, and theta activity to sensorimotor rhythm (SMR

[12-15 Hz]) activity in children with ADHD, with the most significant differences occurring at parietal locations rather than frontal or central locations (Janzen, et al. 1995).

Using elaborate blood flow and glucose metabolism studies, Zametkin (1990) and his colleagues found a decrease in metabolic activity in the frontal cortex of individuals with ADHD, compared to others without ADHD. This indicates that the frontal cortex appears to be underactive, or "underaroused" (Zametkin, et al., 1990). Because specific areas of the frontal lobes control essential mechanisms for inhibition of cortical activity, the condition of being underaroused leaves the brain without adequate means of blocking inappropriate or unimportant sensory input or mental activity (Zametkin, et al., 1990).

Brainstem auditory evoked potentials (BAEP) recorded in studies of attention disordered children have shown prolonged latencies and asymetrical conduction of auditory stimuli in the brainstem implicating a disturbance in the ascending reticular activating systems (ARAS) and its role in auditory processing (Lahat, et al., 1995). This test may be useful in contributing to the diagnosis of ADHD as well as enhancing understanding of the disorder.

The quantitative EEG (QEEG) is a neurometric method sometimes used as a part of the assessment protocol in

ADHD, affectively disordered, and obsessively disordered individuals. A recent study by Suffin and Emory (1995) examined the relationship between location, quantity and coherence (coherence being when two EEG signals at different regions of the cortex maintain a constant phase relationship) of brain wave frequencies, and each disorder. Responses to each of three classes of psychopharmacologic agents: stimulants, antidepressants and anticonvulsant/lithium, were also measured. They found that excess frontal alpha (7.5-12.5 Hz) activity or excess frontal theta (3.5-7.5 Hz) activity, with relative deficits of delta (1.5-3.5 Hz) activity correlated with both ADHD, and with affective disorders. A moderate percentage of inter-hemispheric hypercoherence was seen in each disorder, the remainder were normocoherent. Further examination showed that individuals in the normocoherent groups, regardless of whether ADHD or affectively disordered, who showed excess frontal alpha frequencies responded best to antidepressants; those with excess frontal theta frequencies responded best to stimulants. The hypercoherent groups were resistant to both antidepressants and stimulants regardless of diagnosis, and regardless of presence of excess alpha or excess theta, however they were responsive to the anticonvulsant/lithium class agents (Suffin and Emory, 1995). Another study has shown QEEG to be helpful in differentiating between ADHD, related

disorders, and evaluating response to specific medications (Chabot, et al., 1996).

Neurofeedback

Based on neurometric evidence of disturbances in levels of the brainwave activity referred to in the previous section, Joel Lubar began a series of studies in 1975 of EEG biofeedback, or what is now called "Neurofeedback" (Lubar and Shouse, 1976; Shouse and Lubar 1979; Lubar, 1991, 1993. At first, Lubar trained ADHD children with hyperkenesis to increase SMR activity and to inhibit theta activity. Later he discovered that children who had attention difficulties and problems in learning academic subjects, without associated hyperkenesis were deficient in producing beta activity in addition to producing excess theta activity. He began training these children first to increase SMR activity then to increase beta activity, while at the same time inhibiting theta activity. He found significant and sustained improvements in school performance and psychometric measures in these children following completion of training (Lubar, 1991). Studies by Tansey (1991), Lubar (1991), and Lubar, et al. (1995) have shown improvement in both verbal and performance scores on Wechsler Intelligence Scale for Children - Revised (WISC-R) profiles by ADHD children following treatment with Neurofeedback. Sustained academic improvement of up to ten years after Neurofeedback as

evidenced by improved letter grades are documented by Lubar (1991), Lubar, et al., 1995, and Tansey (1993). Lubar (1997b) cites improvements in several areas using the Conners scale up to ten years after Neurofeedback treatment. The greatest improvements were in general behavior, overall attitude, doing homework, improved grades and family and social relationships (Lubar, 1997b).

It appears that more research is needed to determine the status of Neurofeedback as the most effective and cost-beneficial treatment for ADHD. At this point, the literature looks promising, but the studies are small and may include additional treatment modalities that confound the results. However, new studies are underway which will examine exclusively Neurofeedback effects. S. Othmer (personal communication, June, 1998) is now in the process of collecting data for a multisite study with much larger numbers. Lubar (1997b) is also conducting multisite studies. Neurofeedback or EMG biofeedback used with stimulant medication seems particularly effective (Potashkin and Beckles, 1990). Lee (1991), Lubar (1997b) and Nathan (1992) suggest that the combination of modalities is synergistic, and that the best approach may be individually designed programs utilizing the best combination for that individual.

The drawbacks to wider use of Neurofeedback seem to be lack of up-to-date, sensitive, computerized equipment

capable of filtering and enhancing EEG activity, in an affordable range for clinicians, and the lack of insurance coverage for Neurofeedback treatment of ADHD. Treatment of ADHD with psychopharmaceuticals may be less expensive initially, and relatively safe and effective in some cases. However, they may have undesirable side effects as well as potential for toxic reactions. Another major disadvantage is that the effects of medication last only as long as it is in the body, therefore, it may be best used during initiation of a treatment program to facilitate other modalities.

Given evidence that ADHD often persists into adulthood, a non-invasive therapy with few if any side effects, capable of producing long-term remediation of ADHD may be more desirable than treatment with medications. Neurofeedback has the potential to meet that challenge based on studies by Linden, et al. (1992), Lubar (1991, 1997b), Lubar and Lubar (1984), Lubar, et al. (1995), Lubar and Shouse (1976), Othmer, Othmer and Marks (1992), Othmer and Othmer (1992, 1994), Potashkin and Beckles (1990), Rossiter and LaVaque (1995), and Tansey (1991, 1993). Long-term cost benefits, and cost-effectiveness are part of the potential of Neurofeedback for treatment of ADHD.

Purpose of Study

One purpose of this study was to train children to produce mid-frequency (SMR/beta. 12-18 Hz) EEG activity.

Another purpose of this study was to test the effectiveness of a Neurofeedback training program on objective measures of attention, impulsivity, response time and variability as measured by the T.O.V.A. In addition, it is to test the effectiveness of Neurofeedback on subjective measures of attention, hyperactivity and impulsivity as measured by the Oaks checklist for ADHD.

Hypothesis and Variables

The first hypothesis is that Neurofeedback will result in sustained levels of 12-18 Hz activity over the course of training.

The second hypothesis is that Neurofeedback training will produce significant improvements in T.O.V.A. measures of attention, impulsivity, reaction time, and variability.

The third hypothesis is that Neurofeedback training will produce significant improvement in the Oaks measures of inattention, hyperactivity and impulsivity.

METHOD

Participants

Twenty participants took part in this study. All were patients who sought Neurofeedback for treatment of ADHD. All treatment was performed in a private clinic setting. Participants were selected based on the availability for training and for obtaining pre and post data using the T.O.V.A. and the Oaks Checklist for ADHD. Included in the study were 3 females and 17 males ranging in age from 5 to 15 years, with a mean of 10.5 years. All participants met the following criteria in order to undergo Neurofeedback treatment: (1) Behavior symptoms consistent with DSM-IV(1995) criteria for diagnosis of ADHD. (2) No specific sensory defects or any other comorbid functional or physical illness (e.g., mental retardation, seizure disorders, etc.) that might contribute to, or otherwise be confounded with ADHD. Participants were not excluded if they were on medication to treat ADHD, however parents were cautioned to observe children for an increase in side effects and for signs of overdosage during their participation in the Neurofeedback condition of the study. The study was carried out over a twenty month period beginning in August 1997 and ending in early April of 1999.

This study utilized a two groups mixed factorial experimental design with two different order conditions (2 x 10 x 20). One condition was a Neurofeedback treatment

program (Neurocybernetics, EEG Spectrum, Encino, CA). The second condition was a hands-on mental peak performance computerized training program named Thinkfast (Megabrain Communications and Braintrainment Resources, 1996). The ThinkFast task was used as a credible placebo to control for the computer game feedback and for passage of time waiting for neurofeedback training. These two conditions were used in opposite sequence in each of two groups. Both conditions consisted of 30-minute sessions for a total of twenty sessions in each condition. Sessions were conducted Monday through Saturday over a period of time that ranged from four months to eight months. The goal was to participate in at least two sessions per week. Participants were assigned to one of two groups. After Pretests were conducted, Group 1 received 20 sessions of Neurofeedback, followed by 20 sessions of ThinkFast. Group 2 received 20 sessions of ThinkFast, followed by 20 sessions of Neurofeedback (see Table 1). For the first part of the study, each participant was assigned to either a Neurofeedback treatment condition or ThinkFast. All participants were treated in accordance with the "Ethical standards of Psychologists" (American Psychological Association, 1981).

Table 1

Order of Conditions for Groups 1 and 2

	Pretest	Condition	Posttest 1	Condition	Posttest 2
Group 1	T.O.V.A. & Oaks	Neuro-feedback	T.O.V.A. & Oaks	ThinkFast	T.O.V.A. & Oaks
Group 2	T.O.V.A. & Oaks	ThinkFast	T.O.V.A. & Oaks	Neuro-feedback	T.O.V.A. & Oaks

Apparatus and Materials

Neurofeedback Treatment. Neurofeedback treatment was conducted using Neurocybernetics EEG biofeedback equipment and software (EEG Spectrum/Neurocybernetics, Encino, CA) consisting of a high-gain amplifier (10,000x), an analog to digital converter and two linked computers. The system used an IBM Pentium as a computer interface that displayed the feedback signals to the participant (the Game computer). Another IBM Pentium continuously displayed the raw and filtered EEG signals to the therapist (the Therapist's computer). Thresholds could be updated without interruption of training so that the learning rate could be optimized. The feedback was a pac-man style, video display on a computer monitor, software by Neurocybernetics/EEG Spectrum (Encino, CA), which provided continuous visual and auditory feedback signals to the participant. These instruments were calibrated before this study and remained calibrated throughout the course of the study.

ThinkFast. A mental peak performance training condition was provided using Thinkfast (Megabrain Communications and Braintrainment Resources, 1996), a computerized program consisting of five subprograms (Games) designed to increase mental function in five fundamental capacities. The Thinkfast user's guide describes the series as seeking to improve mental processing speed, efficiency and capacity typically experienced as mental quickness, focus and clarity (Thinkfast User's Guide, 1996).

The goal of Game #1 (Physical Reflexes) is designed to improve physical reflexes, i.e., shorten reaction time. The goal of Game #2 (Perceptual Reflexes) is to improve perceptual reflexes, i.e., increase alertness and visual acuity expressed as perceptual threshold. The goal of Game #3 (Cognitive Reflexes) is to improve cognitive reflexes, i.e., shorten visual information processing time using both visual perceptual and cognitive conditions. The goal of Game #4 (Working Memory Speed) is to improve the decision process that manages access to both short-term and long-term memory. It contains an auditory condition in the form of a tone, which if it sounds, signals the participant to make a response opposite to the correct response. The goal of Game #5 (Working Memory Capacity) is to increase the amount of information that can be consistently and accurately processed at one time (Thinkfast User's Guide, 1996). An example of Game #1 is a square outline, which

briefly flashes on the screen over a 60 second period. Participants were instructed to push the down-arrow key on the computer keyboard as soon as they saw the stimulus. At the end of the game period an evaluation of that game's data was displayed: a scaled measure of speed measured in milliHertz; errors read as a percentage; efficiency read as a percentage; and performance as an adjusted total score. An evaluation of game specific data is provided at the end of each game period. At the end of the game series an adjusted performance score is computed by the game software based on overall performance in each of the five games.

Test of Variables of Attention. The Test of Variables of Attention (T.O.V.A) was administered as part of the intake procedure, and after each phase of the study. The T.O.V.A. is a non-language based, computerized, continuous performance test (CPT) which requires no left-right discrimination, memory or sequencing. Two easily discriminated visual stimuli are presented for 100 milliseconds every two seconds for 22.5 minutes. Scores derived from the T.O.V.A. were measures of Attention (based on errors of omission), Impulsivity (based on errors of commission), Reaction Time (based on mean correct response time), and Variability (based on consistency of response time). The T.O.V.A. has been used to demonstrate significant differences between pretreatment and post-Neurofeedback conditions (Lubar, et al., 1995; Othmer and

Othmer, 1992). Greenberg (1987) reported that there are no test-retest practice effects and participants actually tend to perform more poorly when retested due to boredom.

The Oaks Checklist. The Oaks Checklist for Attention Deficit and Related Disorders (Child/Adolescent Version) (Robert Hill, The Oaks Psychological Services, 1996) was used to assess behavioral changes as rated by parents. The Oaks Checklist is designed to measure the three major characteristics of the DSM-IV (1995) definition of ADHD, and other related behaviors. Subscales used in this study were Inattention (attention deficit), Hyperactivity, and Impulsivity. Forty eight items are included on these subscales.

Procedure

Neurofeedback. Participants were under treatment by, and supervision by a single therapist who had over two years of experience in providing Neurofeedback training for ADHD. This therapist held a bachelor's degree in health care with additional training in Neurofeedback, and certification in Neurofeedback by the Biofeedback Certification Institute of America (B.C.I.A.).

Participants were instructed to let their body relax as much as possible at the same time as keeping their mind alert and focused on the pac-man style feedback game. Theta (4-7Hz) activity and High Beta (22-30Hz) was inhibited, while Beta/SMR (12-18Hz) activity was rewarded.

EEG readings were obtained from bipolar electrode sites situated at C3 with reference electrode at Fpz, and C4 with reference electrode at Pz, based on the international 10/20 placement system (Callaway, 1975). Ground was provided by an earclip electrode. The following physiological responses were monitored during each 30 minute session: high beta activity defined as 22-30 Hz events above threshold; beta activity defined as 15-18 Hz events above threshold over the left hemisphere; SMR activity defined as 12-15 Hz events above threshold over the right hemisphere; and theta activity defined as 4-7 Hz activity above threshold.

Threshold levels were determined, for each participant, from baseline amplitude averages of each of the four levels of activity. Theta thresholds were set at 80% of baseline amplitude average. High beta thresholds were set at 90% of baseline amplitude average. Beta thresholds (left hemisphere) and SMR (right hemisphere) were set at a range of 70% to 90% of baseline amplitude average. Theta thresholds were set at 80% of baseline amplitude average. Averages of all levels were determined during the first two minutes of each session. Thresholds were adjusted when necessary using the therapist computer, to maintain the over-goal percentages of approximately 10% for high beta activity; approximately 80% for beta or SMR activity; and approximately 20% for theta activity.

Protocols for use of left beta reward versus right SMR reward were based on each participant's T.O.V.A. and Oaks Checklist profile. Those participants who had predominately inattentive profiles were given left hemisphere Neurofeedback rewarding increased beta activity and decreased theta activity. Those participants who had predominately impulsive or hyperactive profiles were given right hemisphere Neurofeedback rewarding increased SMR activity and decreased theta activity. Those participants who had mixed profiles (i.e., both inattentive and impulsive or hyperactive) were given left beta reward for half of the session, and right SMR reward for half of the session. Decreasing theta activity was done either by directly inhibiting theta, or by concurrently rewarding beta/SMR activity.

Reward criteria were set so that 50 sampled events occurring in 0.5 second were required in order to receive a reward. Feedback rewards were triggered 0.5 second after the EEG criteria were met. Rewards were both visual and auditory, with a tone and an interactive display on the computer. The display was a maze with dots along the maze in which a yellow, pac-man object progressed, "eating" the dots and accumulating points with each reward criterion reached. The brightness and speed of the pac-man object's progression, and the auditory feedback tone in the form of a "beep" were governed by the levels of EEG amplitudes

relative to pre-selected thresholds. An example of the display was a maze in which the pac-man object would stay bright yellow and advance rapidly along a predetermined course as long as the reward criterion were reached. Theta and high beta activity above threshold would slow or stop pac-man's progression, the object would darken and auditory beeps would stop at the same time. Reward events were defined as production of 15-18 Hz activity or 12-15 Hz activity above threshold in the absence of 4-7 Hz events and 22-30 Hz events above threshold. With these settings for thresholds, participants received an average of 50 rewards per minute. When participants received over 50 rewards per minute consistently, their thresholds were made more difficult. Each session lasted approximately 30 minutes and consisted of 15 periods separated by 10 second intervals during which a bar graph displayed the time and point scores of previous periods to the participant.

ThinkFast. Each participant played Thinkfast for a total of 30 minutes per session. Depending on age and ability each series was repeated between three and six times during a session.

After completing each condition of the study (Neurofeedback treatment or ThinkFast), the T.O.V.A. was administered, and the Oaks checklist was completed by parents.

Test of Variables of Attention. During administration of the T.O.V.A., participants were told to watch the screen and click a thumb control button whenever a black square appeared at the top portion of a an outer white square (target stimulus). If the square appeared at the bottom portion of the outer white square (nontarget stimulus), participants were not to click the button. Results were computed by an in-computer software program designed by the T.O.V.A. Corporation (Universal Attention Disorders, Los Alamitos, CA).

The Oaks. Parents were instructed to rate the child's behavior in the home environment on a scale of 0 to 5 (0 = not present, 5 = very severe) before, and after each phase of the study. Raw scores on each of the subscales were converted into standard scores.

RESULTS

Effectiveness of Neurofeedback Training

A groups by trials ANOVA was conducted on the participant's percentage of time over goal for mid-frequency brainwaves (12-18 Hz). Goals were set at a range of 70% to 90% of baseline amplitude average. The average of the mid-frequency level was determined during the first two minutes of each session. Goal thresholds were adjusted as necessary to maintain over-goal percentages of approximately 80% for mid frequency activity.

The design was a 2 groups x 10 periods x 20 sessions mixed factorial (see Table 1). The analysis revealed one major result. There was a main effect for the 10 training periods, $F(9, 3078) = 2.67$, $p < .01$. This means that, within sessions, groups generally improved in meeting criteria within periods over the 20 sessions. None of the other main effects or interactions were statistically reliable (see Table 2).

Table 2

Mean Percent of Time over Goal

Collapsed over Groups 1 and 2

Periods		Sessions			
Period no.	Mean	Session no.	Mean	Session no.	Mean
1	61.37	1	63.6	11	70.84
2	65.47	2	70.13	12	65.87
3	70.43	3	65.73	13	65.39
4	65.46	4	69.57	14	70.28
5	66.78	5	66.67	15	69.1
6	68.28	6	66.58	16	66.73
7	67.37	7	66.15	17	65.49
8	68.02	8	63.01	18	65.85
9	68.97	9	66.78	19	71.15
10	67.9	10	63.49	20	67.72

Analysis of the T.O.V.A.

A series of two sample comparisons using *F* distributions were done between Group 1 and Group 2 on the Pretest, and Posttest 1, and Posttest 2 scores. Comparison of Pretest scores resulted in two significant findings, for two T.O.V.A. subscales, Impulsivity and Variability. Group 2 scored more favorably than Group 1 on the Impulsivity subscale, F (1, 18) = 6.72, $p < .02$, and Group 2 scored more favorably than Group 1 on the Variability subscale, $F(1, 18) = 12.01$, $p < .002$.

Analyses of Posttest 1 between Group 1 and Group 2 revealed only one comparison that was statistically reliable. Group 2 scored more favorably than Group 1 on the Variability subscale, $F(1, 18) = 6.69$, $p < .02$.

Analyses of Posttest 2 between Group 1 and Group 2 again revealed only one comparison that proved statistically reliable. Group 2 scored more favorably than Group 1 on the Variability subscale, $F(1, 18)) = 3.87$, $p < .07$.

Analyses of Covariance on Posttest 1 and Posttest 2 Scores. Comparing Group 1 and Group 2 on each T.O.V.A subscale involved statistically controlling relevant Pretest subscale scores. The Analysis of Covariance did not yield any significant results with one marginally significant exception for Posttest 1 between Group 1 and Group 2. adjusted mean Impulsivity scores. Group 1 (adjusted mean 90.23) scored more favorably than Group 2 (adjusted mean 73.57). on the Impulsivity subscale.

Analyses of Posttest 1 and Posttest 2 Differences. Comparisons between Posttest 1 and Posttest 2 T.O.V.A. subscale scores were done using correlated t-test procedures. Analyzing Group 1 scores resulted in no significant differences. Analysis of Group 2 scores yielded one significant effect. The Impulsivity scores improved significantly from Posttest 1 to Posttest 2, $t(9) = -5.15$, $p < .01$.

Comparisons Between Pretest and Posttest 1 and Posttest 2

Comparisons between Pretest and Posttest 1 and Posttest 2 T.O.V.A. subscale scores were performed using paired samples *t*-test procedures.

Comparing Pretest scores to Posttest 1 scores in Group 1 yielded no significant effects. Comparing Pretest scores to Posttest 2 scores in Group 1 yielded two significant effects. The Variability subscale scores improved significantly from the Pretest to Posttest 2, $t(9) = 2.16$, $p < .06$. The Impulsivity subscale scores improved from Pretest to Posttest 2, $t(9) = -1.88$ $p < .10$.

Comparing the Pretest scores to the Posttest 1 scores, and the Pretest scores to the Posttest 2 scores in Group 2 yielded no significant effects (see Table 3).

Table 3

Mean T.O.V.A. Scores

	Group 1 (n = 10)			Group 2 (n = 10)		
	Pretest	Post. 1	Post. 2	Pretest	Post. 1	Post. 2
Attention						
M	62.4*	71.3	80.3	86.9*	85.7	87.9
SD	36.85	36.25	27.65	20.72	24.12	24.32
Impulsivity						
M	69.5**	79.8	88.8	92.2**	84	102.6
SD	26.01	32.71	23.86	9.5	14.66	10.75
Reaction Time						
M	73.9	71.7	77	84	83.4	80.9
SD	23.89	28.4	21.1	12.12	24.46	27.5
Variability						
M	46.2***	53.5**	69.9*	81.7***	84.8**	87.8*
SD	31.33	34.41	20.6	8.23	16.77	20.07

Note. Higher scores are more favorable on the T.O.V.A. scale; lower scores are more favorable on the Oaks scale. *$p < .10$. **$p < .05$. ***$p < .01$.

Analysis of the Oaks

A series of two-sample comparisons using F distributions was done between Group 1 and Group 2 on Pretest, and Posttest 1 and Posttest 2 scores. Comparison of Pretest scores resulted in one significant finding.

Analysis of Pretest between Group 1 and Group 2 differed in one Oaks subscale, Impulsivity. Group 2 scored more favorably than Group 1 on the Impulsivity subscale (F (1, 18) = 5.62, $p < .05$).

Analysis of Posttest 1 and Posttest 2 yielded no significant differences between groups on Posttest 1 and Posttest 2 measures.

Analyses of Posttest 1 and Posttest 2 Differences. Comparisons between Posttest 1 and Posttest 2 Oaks subscales scores were performed using correlated t test procedures. Analyzing the Group 1 scores resulted in no significant differences. Analyses of the Group 2 scores yielded one significant effect. The Inattention subscale scores improved significantly from Posttest 1 to Posttest 2 in Group 2, $t(9) = 3.94$, $p < .01$. Marginal differences were found for Group 2 on Hyperactivity and on Impulsivity subscale scores. Group 2 Hyperactivity subscale scores improved, $t(9) = 2.15$, $p < .06$ as did the Impulsivity subscale scores, $t(9) = 2.0$, $p < .08$ from Posttest 1 to Posttest 2.

Comparisons Between Pretest and Posttest 1 and Posttest 2. Comparisons between the Pretest Oaks and Posttest 1 and Posttest 2 Oaks subscale scores were performed using paired samples t-test procedures. Comparing the Pretest and Posttest 1 scores in Group 1 yielded two significant effects. The Hyperactivity subscale scores improved

significantly, $t(9) = 2.54$, $p < .05$ and the Impulsivity subscale scores improved significantly, $\underline{t}(9) = 2.44$, $p < .05$, from the Pretest to Posttest 1. Comparing the Pretest and Posttest 2 scores in Group 1 yielded three significant effects. The Attention subscale scores improved significantly from the Pretest to Posttest 2, $\underline{t}(9) = 2.25$. $p < .05$. The Hyperactivity subscale scores improved marginally from the Pretest to Posttest 2, $\underline{t}(9) = 1.85$, $p < .10$, and the Impulsivity subscale scores improved significantly from the Pretest to Posttest 2, $\underline{t}(9) = 2.57$, $p < .05$.

Comparing the Pretest and Posttest 1 scores in Group 2 yielded no significant effects. Comparing the Pretest and Posttest 2 scores in Group 2 yielded one marginal effect. The Attention subscale scores improved marginally from the Pretest to Posttest 2, $\underline{t}(9) = 2.13$, $p < .10$ (see Table 4).

Table 4

Mean Oaks Scores

	Group 1 (n = 10)			Group 2 (n = 10)		
	Pretest	Post. 1	Post. 2	Pretest	Post. 1	Post. 2
Inattention						
M	54.7	48.9	41.4	51.8	54	41.4
SD	12.21	19.12	12.55	26.08	22.43	23.51
Hyperactivity						
M	57.1*	42.8	42.4	35.9*	38.1	27.3
SD	23.49	17.25	19.61	29.17	29.29	23.93
Impulsivity						
M	66.4**	50.2	47	38**	42	33
SD	17.41	23.86	18.67	33.64	28.82	23.87

Note. Higher scores are more favorable on the T.O.V.A. scale; lower scores are more favorable on the Oaks scale. *$p < .10$. **$p < .05$. ***$p < .01$.

DISCUSSION

Effect of Neurofeedback training

A major purpose of this study (Hypothesis 1) was to demonstrate that children with ADHD could be trained to produce mid-frequency EEG activity within the 12-18 Hz bandwidth (SMR/beta). Evidence of sustained production of criteria levels of SMR/beta EEG activity as measured in percent of time over goal was shown within 10 training periods collapsed across 20 sessions and Group 1 and

Group 2. The particular procedures employed in this study were successful in achieving these results. Participants were given feedback (computer screen game) for the production of SMR/beta activity only when specific voltage amplitudes were crossed within this frequency range. Goal thresholds for SMR/beta EEG were first set at approximately 80% of average baseline amplitude and minimally adjusted as needed to reward time-over-goal percentages of between 70% and 90%. Goal thresholds for theta and high beta were set to inhibit production of these frequencies in excess of 20% for theta, and in excess of 10% for high beta.

Participants were required to consistently maintain or increase production of SMR/beta EEG while inhibiting theta and high beta in order to obtain game rewards. This particular reinforcement contingency procedure has been used successfully by other biofeedback researchers. Bird, Newton, Sheer and Ford (1978) demonstrated that college age students could learn to control 40 Hz EEG activity. Also, Lubar, et al. (1976a, 1976b, 1979) and Lubar (1991, 1997b) demonstrated that children with ADHD could learn to control SMR/beta EEG activity.

It is important to emphasize an important aspect of the procedures that were employed in this study. For each training session, voltage criteria within the SMR/beta bandwidth were adjusted to allow a response rate of 80% of average amplitude. If response rate exceeded or fell below

80%, voltage criteria were adjusted to maintain the response rate within the 70-90% range. This procedural manipulation has implications for response rates within periods and training effects over sessions. Specifically, the EEG data analyzed in this investigation indicate that children successfully maintained the desired levels of SMR/beta activity within training periods. The percent of time that children spent producing SMR/beta was approximately the same at the beginning of the session as at the end. Hence, the children trained in this study maintained criteria levels of SMR/beta throughout the Neurofeedback training sessions. The following will examine the effects of SMR/beta EEG activity on levels of the T.O.V.A. and Oaks tests administered in the present study.

Neurofeedback Effects on T.O.V.A.

Previous studies have linked sustained production of SMR/beta EEG activity to improved T.O.V.A. scores (Linden 1996; Lubar, et al., 1995; Lubar, 1997a; Othmer and Othmer 1992; Rossiter and LaVaque, 1995). Production of SMR/beta EEG has also been correlated to states of inhibition (Lubar, 1997a, 1997b) and increased ability to focus on tasks that are of immediate importance (Linden, 1996; Lubar, et al., 1995; Tansey, 1991

In this study, partial support was found for Hypothesis 2 that Neurofeedback training would improve

performance on the T.O.V.A. subscales of Attention, Impulsivity, Reaction Time and Variability. That is, the Mean T.O.V.A. scores for Impulsivity improved from M = 69.5 on the Pretest to M = 79.8 on Posttest 1 in Group 1. Group 2 showed no improvement on the Impulsivity subscale. The Mean T.O.V.A. scores for Impulsivity improved from a Posttest 1 M = 84 to a M = 102.6 on Posttest 2 in Group 2 following Neurofeedback training. Also, Group 2 scored higher on Posttest 2 following Neurofeedback (M =87.89) than did Group 1 on Posttest 2 following ThinkFast (M = 69.90). No changes were found on Attention and Reaction Time scores (see Table 3).

Neurofeedback Effects on the Oaks

The literature suggests that Neurofeedback training is positively correlated with improvement on behavioral measures as well as improvement in T.O.V.A. scores (Linden, 1996; Lubar, et al., 1995; Lubar, 1997b; Rossiter and LaVaque, 1995). Again, the production of SMR/beta has been correlated to states of inhibition (Lubar, 1997a, 1997b) which are associated with better impulse control. This is reflected in subjective behavioral rating scales such as the Oaks checklist for ADHD. The Oaks is a subjective measure of ADHD characteristics similar to the McCarney Attention Deficit Disorders Evaluation Scale (ADDES) subscales used by Lubar (1995) and favored by Barkley and his colleagues (Lubar, 1997b).

Partial support was found in this study for the Hypothesis 3 that Neurofeedback would improve scores on the Oaks subscales. The subscales used in this study were Inattention, Hyperactivity and Impulsivity. The children were rated on the Oaks scale by their parents. These three subscales corresponded closely to those of the T.O.V.A. used in this study as an objective measure of ADHD characteristics. Mean Oaks scores for Hyperactivity improved from $\underline{M}$ = 57.10 on the Pretest to $\underline{M}$ = 42.80 on Posttest 1 in Group 1 following Neurofeedback. Similarly, Group 1's Impulsivity scores improved from $\underline{M}$ = 66.40 on the Pretest to $\underline{M}$ =50.20 on Posttest 1. Group 2 showed no comparable improvement following ThinkFast. The Oaks scores for Attention, Hyperactivity and Impulsivity improved from Posttest 1 to Posttest 2 in Group 2 after Neurofeedback. No comparable effect occurred in Group 1 after ThinkFast (see Table 4).

ThinkFast Effects

Of interest in this study was the effect of ThinkFast on both the T.O.V.A. and the Oaks scores. ThinkFast was included in this study as a credible placebo for the purposes of controlling for 1) attention effects, 2) the passage of time while children waited for Neurofeedback, and 3) computer game practice effects. The Posttest means suggest that ThinkFast may have had a treatment effect. That is, Group 1 showed within group improvements on the

T.O.V.A. scores for Impulsivity and Variability on Posttest 2, compared to Posttest 1, following ThinkFast. A similar effect was found on all three Oaks subscales, Attention, Hyperactivity and Impulsivity.

General Discussion

Lack of more robust results appears to be partly due to the large difference in Pretest means between groups (see Table 3 and 4). For example, there was a 27% difference between Group 1 and Group 2 on the Pretest T.O.V.A. means, and a difference of 30% on the Oaks means. Because of this large group difference, between-groups comparisons on Posttests did not accurately reflect differences. The higher Pretest means, especially on the T.O.V.A., for Group 2 made it difficult for Group 1 to show improvement on the Posttest scores. In addition, high within-group variability combined with the small number of participants (n=20) also adversely affected the study's statistical power.

Frequency and consistency of training are known to be important in any learning situation. Neurofeedback is no exception. Lubar, et al. (1995) conducted daily Neurofeedback sessions of one hour for their study. Others have used two to three sessions per week (Linden, 1996; Lubar, 1997b; Othmer and Othmer, 1992). Although the goal of this study was to conduct training sessions twice weekly, in reality, it was not always consistent.

Because of technical considerations, the first 10 applicants accepted into the study were assigned to Group 1. Most of Group 1 participants were required to finish their part in the study before the second 10 participants started their part. This precluded matching participants between groups or randomly assigning participants to the groups. In addition, Group 1 took much longer than Group 2 to complete the study. Absences due to Thanksgiving and Christmas-New Years holidays for Group 1 also had an effect on the continuity of training.

Lubar (1997b) and others (Linden, 1996; Othmer, et al., 1992; Tansey, 1991) report the best results on T.O.V.A. and other measures when Neurofeedback was carried out over 30 to 45 sessions. Unfortunately, the time constraints for this study limited Neurofeedback training to just 20 sessions.

In a study of this design, it is important to match groups as closely as possible. Specifically, this means that every attempt should be made to approximate Pretest scores between groups so that the groups are comparable at the beginning. It would also be preferable to run both groups in tandem rather than sequentially, as was done in this study. Changing the design in this manner would eliminate some of the problems in terms of discovering robust results.

Future research should include more studies which investigate the correlation between EEG changes and changes in behavioral measures. Lubar (1997b), and Othmer and Othmer (1992) are among the few who examine both EEG and its effect on behavioral measures. It is important to establish this connection between Neurofeedback and cognitive and behavioral changes to further legitimatize Neurofeedback as an effective treatment for ADHD. In addition, there is much to be learned about its use for other conditions known to have a neurological basis.

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